Transient Ischemic Attack and Central Retinal Artery Occlusion

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KEYWORDS

• TIA • CRAO • ABCD score • Transient ischemic attack

KEY POINTS

- A transient ischemic attack (TIA) is now defined by tissue-based criteria rather than time, and confirmation requires negative MRI findings.
- Most TIA patients are asymptomatic at presentation, as symptoms typically resolve within an hour.
- TIA confers a high risk of subsequent stroke, making early recognition and intervention critical.
- Central retinal artery occlusion is an ophthalmologic emergency that requires urgent diagnosis and treatment to prevent permanent vision loss.

INTRODUCTION

A transient ischemic attack (TIA) is an acute episode of neurologic dysfunction involving the brain, eye, or spinal cord, caused by focal ischemia in a vascular territory that resolves quickly without evidence of acute infarction on diffusion-weighted imaging (DWI). The transient nature of TIAs and their limited recognition by the general public contribute to challenges in accurately estimating their annual incidence. Underdiagnosis and failure to seek medical attention further obscure the true prevalence of the condition. Nonetheless, approximately 240,000 TIAs are estimated to occur annually in the United States.[1] The occurrence of TIA has been shown to increase with advancing age and is more common among Black Americans, who are at 1.4 times greater risk compared to White Americans.[1] Although TIA symptoms resolve spontaneously, prompt identification and treatment are crucial, as the risk of stroke within 90 days of a TIA is up to 17.4%.[2]

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Emerg Med Clin N Am 44 (2026) 71–83

https://doi.org/10.1016/j.emc.2025.08.005 **emed.theclinics.com**

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Abbreviations	
ACEP	American College of Emergency Physicians
BNP	B-type natriuretic peptide
CRAO	central retinal artery occlusion
CTA	computed tomography angiography
CUS	carotid ultrasound
DAPT	dual antiplatelet therapy
ED	emergency department
IAT	intra-arterial thrombolysis
LVO	large vessel occlusion
MRA	magnetic resonance angiography
NA	nonarteritic
TIA	transient ischemic attack
tPA	tissue plasminogen activator

From Classic to New: the Definition of Transient Ischemic Attack

The definition of TIA emerged in the 1950s, initially using a time-based framework. TIA was described as an acute, focal neurologic deficit affecting the brain, eye, or spinal cord, attributed to a specific vascular territory, which typically resolves within 24 hours of symptom onset, with an emphasis on the rapid resolution within this timeframe.[3] During this period, some experts advocated limiting the symptom duration to a maximum of 1 hour to differentiate TIA from stroke, as most data suggested symptoms typically lasted around 10 minutes.[4,5] However, in 1964, Marshall proposed extending the duration to 24 hours despite evidence showing that 75% of his patients experienced symptoms lasting less than 1 hour.[6] In 1975, the National Institutes of Health (NIH) revised its classification document and adopted the arbitrary 24-h time-based definition.[7]

In 2009, the American Heart Association (AHA) introduced a *tissue-based* definition driven by advancements in imaging that improved the detection of cerebral infarction.[8] Studies showed that up to 33% to 50% of patients diagnosed with TIA using the classic time-based criteria had evidence of cerebral infarction, challenging the assumption that symptom resolution within 24 hours indicated no brain injury.[3,8] Additionally, as stroke treatments became more common, the time-based definition raised concerns about excluding patients who could benefit from therapy. A 1995 National Institute of Neurological Disorders and Stroke (NINDS) study demonstrated that tissue plasminogen activator (tPA) improved outcomes in patients presenting with stroke-like deficits when treated within 3 hours of symptom onset, while only 2% of patients who received the placebo experienced full resolution at 24 hours.[9] This highlighted the risk of delaying treatment based on an outdated 24-h threshold, which was also inconsistent with findings that most TIA symptoms resolve within 1 hour. These arguments ultimately helped eliminate time as a factor in diagnosis, making tissue injury the pivotal element when defining TIA.

Presentation

Patients with a TIA often present to the emergency department (ED) reporting symptoms resembling those of an acute ischemic stroke. These may include the sudden onset of unilateral weakness, sensory deficits, changes in speech, vertigo, or vision loss. Unilateral weakness can range from complete loss of function to nondermatomal weakness of an extremity. Sensory changes may present as either complete loss of sensation or paresthesias. Speech disturbances can manifest as speech arrest, word-finding difficulty, or slurred speech. Vision loss is typically painless and may involve 1 eye (amaurosis fugax) or a visual field deficit, such as a quadrantanopia. However, as previously noted, TIA symptoms typically resolve quickly, and patients return to their normal neurologic baseline.

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Emergency medicine physicians must distinguish if the patient is experiencing a true TIA or a mimic, which can be challenging. TIA mimics (**Box 1**), such as metabolic disturbances, drug toxicities, seizures, and psychiatric disorders, can present similarly to stroke. In a single-center prospective cohort study aiming to find features that could assist in distinguishing a true TIA versus a TIA mimic, it was found that epileptic seizures and migraine attacks were the most common TIA mimics, at 43.7% and 23.6% respectively, amongst a cohort of 303 patients presenting with concern for TIA.[10] The study showed that patients with unilateral paresis were more likely to have a true TIA than patients without unilateral paresis. Further, it identified that patients with memory loss, headache, or blurred vision had increased odds of having a TIA mimic, with odds ratios of 9.17, 3.71, and 2.48, respectively.[10] Identifying these mimics is critical to avoid unnecessary imaging, prolonged hospitalization, or inappropriate interventions. In addition, EM physicians should assess key risk factors (**Box 2**), including hypertension, atherosclerosis, hyperlipidemia, tobacco use, diabetes mellitus, hypercoagulable states, and atrial fibrillation. Which can increase or decrease the likelihood of someone having a TIA/stroke or it can provide pertinent details to identify possible mimickers.

Management of transient ischemic attack in the emergency department

Emergency medicine physicians should think beyond just imaging, as identifying the underlying cause of the transient neurologic symptoms is essential for optimal management. The trial of Org 10172 in acute stroke treament (TOAST) criteria (**Box 3**) provide a structured approach, categorizing ischemic stroke/TIA mechanisms into large artery atherosclerosis, cardioembolism, small vessel disease, stroke of other determined etiology, and stroke of undetermined etiology. This framework can guide the diagnostic workup of EM physicians, including vascular imaging, cardiac evaluation, laboratory testing, and medical treatment.[11]

Neuroimaging

It is well known that obtaining neuroimaging in the ED is pivotal when evaluating patients with concerns about a TIA. With the shift from a time-based to a tissue-based definition,

Box 1
Common transient ischemic attack mimics encountered in the emergency department

TIA Mimics

Metabolic conditions

Drug toxicity

Seizure

Complex migraine

Psychogenic/functional disorder

Syncope

Infection

Peripheral vertigo

Brain tumors

Adapted from Amort M, Fluri F, Schäfer J, et al. Transient ischemic attack versus transient ischemic attack mimics: frequency, clinical characteristics and outcome. Cerebrovasc Dis 2011;32(1):57–64. https://doi.org/10.1159/000327034.

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Box 2
Common comorbid conditions and risk factors for transient ischemic attack (TIA)

Risk Factors for TIA

Hypertension

Hyperlipidemia

Diabetes Mellitus

Tobacco use

Atrial Fibrillation/Atrial Flutter

Hypercoagulable disorders

Cancer

Previous TIA or Stroke

Atherosclerotic disease

Vasculopathy

Cardiomyopathy

Valvular disease

distinguishing TIA from an actual ischemic stroke is crucial. The gold standard for ruling out stroke is an MRI with diffusion-DWI, the most sensitive and specific modality. However, a noncontrast head CT is often the initial step in the ED due to its speed and ability to identify an intraaxial mimic, such as mass or hemorrhage. While MRI should be obtained as soon as possible, logistical barriers in some settings highlight the need for expedited follow-up with a TIA clinic or neurologist.[12]

After the initial noncontrast head CT, neurovascular imaging is recommended to evaluate possible vascular causes for a TIA, as carotid artery stenosis is a common finding that can lead to atherothrombosis or reduced perfusion, resulting in acute neurologic deficits. While digital subtraction angiography is considered the gold standard, its invasiveness, cost and procedural time make it less favorable as a first-line option, especially since other imaging modalities offer comparable accuracy.[13]

Three noninvasive imaging techniques—carotid ultrasound (CUS), computed tomography angiography (CTA), and magnetic resonance angiography (MRA)—are commonly

Box 3
This table summarizes the TOAST (trial of Org 10172 in Acute stroke Treament) criteria used to classify ischemic stroke etiology

TOAST Criteria

Large artery atherosclerosis

Cardioembolism

Small vessel disease

Stroke of other determined etiology

Stroke of undetermined etiology

Adapted from Low molecular weight heparinoid, ORG 10172 (danaparoid), and outcome after acute ischemic stroke: a randomized controlled trial. The Publications Committee for the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) Investigators. JAMA 1998;279(16):1265–72.

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used to assess the degree of carotid stenosis, each with distinct advantages and limitations. CUS is widely accessible but highly operator and facility-dependent. Conversely, CTA provides more precise and rapid imaging, allowing for the assessment of the aortic arch, external and internal carotid arteries, and intracranial vessels.[14,15] However, it may not be suitable for patients with renal impairment due to the use of contrast. Additionally, metallic artifacts and contrast allergies can limit its utility. MRA is an excellent alternative for patients who are unable to undergo CUS or CTA, offering high sensitivity and specificity without the use of radiation or iodinated contrast. Nevertheless, its availability is limited in some hospitals, and it is contraindicated in patients with specific implanted medical devices or retained metallic fragments.[12,16] CT perfusion scan can be obtained if the patient is having stuttering symptoms, as this could be indicative of a subocclusive thrombosis, which poses a high risk for stroke.

Cardiac Assessment

Initial workup in the ED should include a cardiac evaluation, as 6% to 31% of TIAs are caused by a cardioembolic source.[17] The assessment typically includes obtaining laboratories such as troponin and B-type natriuretic peptide (BNP), a 12-lead ECG, 24-h telemetry, and echocardiography to identify high-risk features that may lead to embolism; specific workups may vary depending on the physician's preference or patient risk factors. These include, but are not limited to, arrhythmias, valvular disease, reduced left ventricular function, cardiomyopathies, and cardiac masses.[16]

Laboratory Studies

There is significant interest in identifying biomarkers that could aid in the early and accurate detection of TIA or stroke; however, none have been fully validated. Nonetheless, EM physicians should obtain laboratories to assess for potential underlying etiologies and guide secondary prevention strategies. Laboratory tests include a complete blood count, chemistry panel, coagulation studies, inflammatory markers, lipid panel, hemoglobin A1c, troponin, and BNP. Laboratories are also helpful when trying to rule out possible TIA mimics, such as recrudescence in a patient with a previous stroke that can be exacerbated by infection or metabolic derangements. A significant emphasis is on obtaining glucose levels in patients, as hypoglycemia is a common mimicker of TIA or stroke. Nonetheless, additional tests may be ordered based on consultant recommendations or concerns about specific alternative diagnoses[16]

Medical Therapy

EM physicians should be familiar with the treatment of TIA, as consultation with a stroke physician or general neurologist may not be an immediate option. Nonetheless, neurology specialists comfortable with managing patients with TIA should be consulted when selecting medical therapy and to establish continued management. In general, the EM physician should understand that the management of TIA focuses on controlling blood pressure, managing comorbid conditions that increase the risk for stroke, and initiating antiplatelet or anticoagulation therapy depending on stroke etiology and patient factors.

One of the most extensively studied aspects of medical management in TIA and minor stroke is antiplatelet therapy. Dual antiplatelet therapy (DAPT) is a cornerstone of treatment of high-risk (ABCD2 score $\geq$4) TIA, as recommended by the AHA and supported by multiple randomized controlled trials and meta-analyses. Studies have shown that DAPT is superior to aspirin monotherapy in patients with high-risk TIA, demonstrating a greater reduction in stroke recurrence within 90 days and lower rates of major adverse cardiac events. While DAPT carries a slightly increased risk of major

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bleeding events, its hemorrhagic stroke risk remains comparable to aspirin alone. Notably, the bleeding risk is further reduced when DAPT is limited to 21 days of treatment. However, it is generally recommended that patients with low-risk (ABCD2 score <4) TIA's should be treated with aspirin monotherapy.[16,18–21]

Disposition and scoring tools

One of the most challenging tasks for EM physicians is deciding the disposition for patients with TIA. Clinicians must consider several factors when choosing the disposition for their patient, weighing their patients' risk of stroke shortly after their TIA. To help provide a solution, researchers developed a scoring system that predicts stroke risk in these patients. In 2005, the ABCD score was introduced as a tool to identify individuals at high risk of stroke within 7 days of a TIA, helping guide decisions on whether they needed inpatient admission or could be safely discharged.[22] The score incorporated 4 key factors: *A*ge, *B*lood pressure, *C*linical features, and *D*uration of symptoms. Early studies were promising, offering a straightforward and accessible method for stratifying risk. But over time, it became clear that the score didn't perform as well as hoped. Patients labeled as low-risk or moderate-risk were often at higher risk than predicted, largely because the score wasn't adequately validated and left out key risk factors like diabetes, atrial fibrillation, and carotid stenosis. These limitations led to the development of newer scoring systems aimed at improving risk stratification and more accurately predicting stroke after a TIA.[23–25]

Despite ongoing efforts to refine risk stratification, no clinical decision tool has reliably stratified TIA patients to determine who can be safely discharged from the ED. In 2016, the American College of Emergency Physicians (ACEP) reviewed the available evidence. It concluded that no existing tool, including the widely used ABCD2 score, was sufficiently accurate to guide disposition decisions independently. While the ABCD2 score (**Table 1**), endorsed by the AHA in 2009, remains helpful in estimating short-term stroke risk, its limitations are well documented. The more recently validated Canadian TIA

Table 1
ABCD2 Score

ABCD2 Score	Components	Scoring System
Age	≥60 y of age	1
Blood pressure	≥140/90	1
Clinical Features	Unilateral weakness	2
	Or	
	Speech Disturbance without weakness	1
Duration	≥ 60 min	2
	Or	
	10 min to 59 min	1
Diabetes	History of diabetes	1

Score: Patients with higher scores have a higher risk of recurrent TIA or stroke.
High: 6 to 7.
Moderate: 4 to 5.
Low: 0 to 3.
Adapted from Johnston SC, Rothwell PM, Nguyen-Huynh MN, et al. Validation and refinement of scores to predict very early stroke risk after transient ischaemic attack. Lancet 2007;369 (9558):283–92. https://doi.org/10.1016/S0140-6736(07)60150-0.

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Score (Table 2) offers improved accuracy over both the ABCD2 and ABCD2i scores, as it incorporates additional clinical variables to better stratify risk. However, its complexity may limit widespread adoption in time-sensitive ED settings.[26,27]

Ultimately, disposition decisions in the ED generally fall into 3 categories: admission to the inpatient service, placement in an observation unit, or discharge home with expedited follow-up through a TIA clinic or neurologist within 48 hours. When determining whether a patient needs to be admitted or can be safely observed, clinicians should consider several key factors. These include an ABCD2 score greater than 4 (indicating higher short-term stroke risk), neurovascular imaging showing significant carotid stenosis, ECG findings of atrial fibrillation or high-risk findings on echocardiography, crescendo or dual TIA symptoms, and the patient's ability to access outpatient neurology care and initiate secondary prevention. Nonetheless, the ED physician's clinical judgment remains essential.

While this list is not exhaustive, it reflects common high-yield considerations. Patients with a low ABCD2 score, a single transient episode, unremarkable cardiac and neurovascular evaluations, and reliable follow-up may be reasonable candidates for discharge with close outpatient monitoring.[25,26,28]

Future directions

Future directions in TIA management in the ED involve integrating bedside point-of-care imaging, biomarkers, and improved risk-stratification tools. The ACEP clinical policy on TIA discussed the potential role of emergency physicians using point-of-care ultrasound

Table 2
Canadian transient ischemic attack score

Clinical Findings	Scores
First TIA of Life	+2
Symptom duration ≥ 10 min	+2
History of carotid stenosis	+2
Current antiplatelet use	+3
History of gait disturbance	+1
History of unilateral weakness	+1
History of vertigo	−3
Presenting diastolic blood pressure ≥ 110 mm Hg	+3
History or examination with dysarthria or aphasia	+1
ED investigations	
Atrial fibrillation on ECG	+2
New or Old infarction on CT	+1
Platelet count ≥ 400,000/L/L	+2
Glucose ≥15 mmol/L	+3

Risk of stroke or carotid revascularization in 7 d.
Low Risk (0.5%): −3 to 3.
Medium Risk (2.3%): 4 to 8.
High Risk (5.9%): ≥ 9.
Adapted from Johnston SC, Rothwell PM, Nguyen-Huynh MN, et al. Validation and refinement of scores to predict very early stroke risk after transient ischaemic attack. Lancet 2007;369 (9558):283–92. https://doi.org/10.1016/S0140-6736(07)60150-0.

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to assess for severe carotid stenosis, emphasizing the need to determine its accuracy in this setting. A supporting study found that when experts used handheld ultrasound devices, the sensitivity and specificity for detecting carotid stenosis of more than 50% were 92% and 93%, respectively.[29] Given emergency physicians' proficiency in bedside ultrasonography, incorporating this tool could be particularly valuable in resource-limited settings while also expediting care.

Beyond imaging, biomarkers such as BNP, microRNAs, and inflammatory markers show promise in identifying stroke risk and aiding early diagnosis of TIA, although further validation is needed before clinical adoption.[30]

Risk stratification scales remain useful, and ongoing research may yield a tool that reliably identifies patients safe for ED discharge. The Canadian TIA Score, for instance, has demonstrated greater accuracy than the ABCD and ABCD2 scores, though its complexity and novelty have slowed widespread adoption.[26]

TIA management has advanced significantly, and as scoring systems, biomarkers, and imaging evolve, they will further improve risk assessment and patient outcomes.

Central retinal artery occlusion

Central retinal artery occlusion (CRAO) is an ophthalmologic emergency. It is defined as cessation of blood flow through the central retinal artery by thromboembolism or vasospasm with or without retinal ischemia.[31] CRAO is classified as arteritic (inflammation of medial and large vessels, 5% of cases) and nonarteritic (ischemic, 95% of cases.)[32] The most common cause of arteritic CRAO is Giant Cell Arteritis. In nonarteritic cases (NA-CRAO), the most common cause is embolism from the ipsilateral carotid artery or the heart. This section will focus on NA-CRAO.

Patients with CRAO typically present with acute, painless monocular vision loss, often accompanied by a reduction in both visual acuity and peripheral vision. Some patients may experience a transient course, as seen in NA-CRAO, with symptoms that can persist for minutes to hours. On physical examination, EM physicians often observe a deficit in finger counting, indicative of peripheral field loss, as well as central vision impairment consistent with a central scotoma. A relative afferent pupillary defect is also commonly identified using the swinging flashlight test.

Fundoscopic examination of NA CRAO reveals hypoperfusion of the retina and retinal edema, with signs of retinal infarction. This includes retinal opacity in the posterior pole, a cherry red spot, optic disc edema, and pallor.[33] Fluorescein angiography often shows a central scotoma and poor retinal circulation.[32] This can be distinguished from *transient* NA CRAO, which typically shows similar fundoscopic findings but demonstrates normal retinal circulation on fluorescein angiography.[32]

Given the rapid onset, EM physicians should have a low threshold to activate the stroke pathway. Rapid diagnosis and treatment are paramount. An ophthalmology consultation for a formal fundoscopic examination is necessary to exclude mimics. This includes retinal detachment, optic neuropathy, vitreous and retinal hemorrhage. Noncontrast CT of the head and basic coagulopathy laboratories should be evaluated. Erythrocyte sedimentation rate (ESR) and c-reactive protein (CRP) will help to discriminate between arteritic and nonarteritic causes. Although part of the stroke workup to identify a large vessel occlusion, CT angiography of the head and neck may not identify a CRAO due to the small size and distal location of the occluded vessel (**Fig. 1**). However, finding a stenotic or occluded ipsilateral carotid artery is common (**Fig. 2**). If presentation is within 4.5 hours, administering intravenous thrombolytics (tissue plasminogen activator [tPA] or tenecteplase [TNK]) is the standard of care. Thrombolytics improve long-term visual outcomes and are likely safe; intracerebral hemorrhage is rare.[34] One meta-analysis of 396 patients with NA CRAO showed that fibrinolysis

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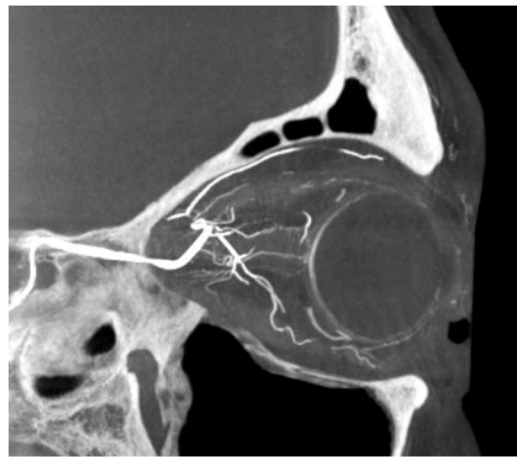

Fig. 1. Dyna thin MIP demonstrating central retinal artery. (*From* https://neuroangio.org/; and *Image Courtesy of* Dr Eytan Raz, NYU Langone Hospital.)

was beneficial at 4.5 hours or earlier compared with the natural history group, demonstrating an absolute risk reduction of 32.3% and a number needed to treat of 4.[34]

For patients who present outside of the 4.5-h window for thrombolytics or have other contraindications, neurointerventional surgery is an option. If CTA identifies a proximal large vessel occlusion (LVO), thrombectomy can be performed. If no LVO is identified, intra-arterial thrombolysis (IAT) into the ophthalmic artery can be attempted. Risks of IAT include arterial dissection, catheter-induced spasm, and further embolizing plaque. IAT in CRAO has been studied with mixed results. In the EAGLE study, IAT was performed on patients up to 24 hours after presentation but did not demonstrate superiority to conservative therapy.[35] However, in patients within the 6-h window, IAT may have benefits.[36]

Fig. 2. MRI, MRA demonstrating a left internal carotid artery occlusion. (*From* https://neuroangio.org/; and *Image Courtesy of* Dr Eytan Raz NYU Langone Hospital.)

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Multiple ongoing trials are reviewing the efficacy of early thrombolysis for NA CRAO. THEIA is a phase III randomized blinded multicenter study examining the efficacy and safety of IV tPA in CRAO (URL: ClinicalTrials.gov. Unique identifier: NCT03197194), REVISION (Early Reperfusion therapy with intravenous alteplase for recovery of vision in acute central retinal artery), investigates IV tPA within 4.5 hours of monocular vision loss due to acute CRAO. Finally, (Tenecteplase in Central Retinal Artery Occlusion Study; URL: ClinicalTrials.gov, Unique identifier: NCT04526951) studies the efficacy of Tenecteplase in CRAO.

Nonthrombolytic therapies for NA-CRAO have been explored but with limited success. Supportive measures such as ocular massage, hyperbaric oxygen therapy, and vasodilation with sublingual isosorbide dinitrate or systemic pentoxifylline have been attempted. Strategies to enhance intraocular perfusion by lowering intraocular pressure, including rebreathing expired carbon dioxide, IV acetazolamide, and mannitol, have not demonstrated a clear benefit.[37,38] Case reports suggest potential improvement in visual acuity with prolonged anticoagulation using heparin or warfarin. While systemic steroids remain the standard treatment of arteritic CRAO, their role in NA-CRAO is unproven, though they may theoretically reduce endothelial edema.

Secondary prevention is essential in reducing the risk of recurrence. Key measures include optimizing blood pressure and lipid management, moderating alcohol consumption, implementing lifestyle modifications, and initiating antiplatelet therapy when appropriate.

CRAO is an ophthalmologic emergency requiring a multidisciplinary approach involving EM physicians, neurologists, and ophthalmologists. Prompt recognition is essential—physicians must maintain a high index of suspicion, systematically exclude mimics, and initiate the stroke pathway without delay. IV thrombolysis remains the standard of care, with neuro interventional surgery considered in select cases. As ongoing clinical trials continue to refine treatment approaches, one principle remains clear: timely intervention is critical to preserve vision.

CLINICS CARE POINTS

- MRI with diffusion-weighted imaging (DWI) is the gold standard for evaluating suspected TIA, defined as transient neurologic deficits without evidence of ischemia on DWI.
- Always assess for transient ischemic attack (TIA) mimics—such as seizures, migraines, or metabolic disturbances—to avoid misdiagnosis and unnecessary treatment.
- Patients with high-risk TIAs (ABCD2 score ≥ 4) should be started on dual antiplatelet therapy within 24 hours of symptom onset.
- While the ABCD2 score aids in early risk stratification, it should not be used in isolation to guide discharge decisions.
- Central retinal artery occlusion (CRAO) is a neuro-ophthalmologic emergency that warrants urgent evaluation and multidisciplinary management, including prompt consultation with ophthalmology and neurology. It should be addressed similarly to acute ischemic stroke.
- EM physicians must assess for other vision-threatening conditions that mimic CRAO, such as giant cell arteritis, as delayed recognition may result in irreversible vision loss.

DISCLOSURE

The authors have nothing to disclose.

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